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# Clinical utility of ocular vestibular evoked myogenic potentials (oVEMPs)

Konrad P. Weber, MD[1,2] ;Sally M. Rosengren, PhD[3,4]

[1]Department of Neurology, University Hospital Zurich, Frauenklinikstrasse 26, CH-8091 Zurich, Switzerland.
[2]Department of Ophthalmology, University Hospital Zurich, Frauenklinikstrasse 24, CH-8091 Zurich, Switzerland.
[3]Neurology Department, Royal Prince Alfred Hospital, Missenden Rd, Sydney NSW 2050, Australia
[4]Central Clinical School, University of Sydney, Sydney, NSW 2006, Australia

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*Corresponding author:*
Konrad P. Weber, MD
Department of Neurology
Frauenklinikstrasse 26
CH-8091 Zurich
Switzerland
Email: konrad.weber@usz.ch

Sally Rosengren, PhD
Royal Prince Alfred Hospital
Neurology Department
Level 8
Missenden Rd
Camperdown NSW 2050
+61295157565
Email: sally@srosengren.org

**Abstract**
Over the last years, vestibular-evoked myogenic potentials (VEMPs) have been established as clinical tests of otolith function. Complementary to the cervical VEMPs, which assess mainly saccular function, ocular VEMPs (oVEMPs) test predominantly utricular otolith function. oVEMPs are elicited either with air-conducted (AC) sound or bone-conducted (BC) skull vibration and are recorded from beneath the eyes during up-gaze. They assess the vestibulo-ocular reflex and are a crossed excitatory response originating from the inferior oblique eye muscle. Enlarged oVEMPs have proven to be sensitive for screening of superior canal dehiscence, while absent oVEMPs indicate a loss of superior vestibular nerve otolith function, often seen in vestibular neuritis or vestibular Schwannoma.

**Keywords [4-6]**
ocular vestibular evoked myogenic potential; oVEMP; vestibulo-ocular reflex; otolith; utricle.

## Introduction

*What are oVEMPs?*

Vestibular evoked myogenic potentials (VEMPs) are vestibular-dependent reflexes thought to originate in the otolith organs - the saccule and utricle - and recorded from the extraocular and cervical muscles. To elicit these reflexes, the vestibular organs are stimulated with short bursts of loud air-conducted (AC) sound or bone-conducted (BC) skull vibration and muscle activity is recorded from surface electrodes placed over or near the respective muscles. Cervical VEMPs recorded from the neck muscles (cVEMPs) were first described 20 years ago [1] and have since become a widely used test of saccular otolith function. The ocular VEMP (oVEMP) is a more recently-described reflex [2,3], which is thought to reflect predominantly utricular otolith function and can be understood as part of the vestibulo-ocular reflex (VOR). The VOR eye movements evoked by vestibular stimulation with sound or vibration are very small, but are produced by highly synchronous bursts of muscle activity which can be recorded by surface electrodes placed near the eyes [3,4].

Although a variety of oVEMPs can be recorded from around the eyes under different stimulation and recording conditions, the most clinically-relevant oVEMP is recorded from beneath the eyes during up-gaze. Under these conditions, the oVEMP is large [5,6] and highly lateralized, being larger on the side contralateral to the stimulated ear [7,8], important factors which contribute to its clinical utility. The lateralization of the reflex is significant as the largest oVEMPs are usually recorded using BC stimulation of the skull, which activates the vestibular receptors in both ears simultaneously. The contralateral dominance means that in patients with unilateral vestibular loss the oVEMP will be absent or attenuated on the side opposite the lesion, revealing the side of the deficit [8]. oVEMPs evoked by AC stimulation are also larger and more prevalent on the contralateral side.

The oVEMP consists of a series of waves, beginning with a negativity with peak latency of about 10 ms (n10) and a positivity at ~15 ms (p15) (Figure 1). Needle recordings from within the inferior extraocular muscles have shown that the n10-p15 response originates in the inferior oblique (IO) muscle [9]. Weber et al. recently recorded single motor unit activity from the IO muscle during oVEMP stimulation and showed an excitation at about 10 ms, similar to the clinical oVEMP recorded during up-gaze, indicating that the primary origin of the oVEMP is the IO muscle [9,10].

oVEMPs are considered a test of utricular function for two main reasons. First, evidence from animal studies suggests that otolith afferents are preferentially activated by sound and vibration [11,12]. Second, patients with vestibular neuritis that appears to selectively affect the superior vestibular nerve typically have absent oVEMPs, suggesting that the reflex depends upon this nerve portion [13-16]. The superior vestibular nerve contains all utricular afferents, but only a small portion of saccular afferents. Together this evidence implies that oVEMPs reflect predominantly utricular function. Contributions from semicircular canal afferents cannot be ruled out [12], but are assumed to be small, and a role for the saccule in oVEMPs in some conditions has not been excluded to date.

oVEMPs can be evoked by a variety of AC and BC stimuli, such as square waves, sine waves and tendon hammer taps. The advantages of BC stimulation are that it does not rely on the middle ear for transmission to the vestibule and it produces larger and more reliable oVEMPs. oVEMPs can be recorded in patients of all ages, including the elderly [17] and children [18], but become smaller and less prevalent with increasing age [19-23]. For oVEMPs evoked by

BC stimulation, the effects of age are greater for square waves and sine waves [24,17,20,23] compared to tendon hammer taps and low-frequency pulses [20,25]. Age is a greater problem for oVEMPs evoked by AC sound, as they are generally smaller than those evoked by BC stimulation. The decline with age leads to a high rate of absent responses in older normal subjects [21,22], reducing the clinical utility of the reflex when evoked by AC sound. The false positive rate for BC oVEMPs is much lower, although skull vibration can be variable and does not always produce robust responses. For this reason BC vibration is probably the better stimulus, though it may be less sensitive in detecting superior canal dehiscence (SCD) [26]. Due to the systematic effects of age and stimulus parameters on oVEMP amplitudes and latencies, patients should ideally be compared to normal age-matched controls tested under the same stimulus and recording conditions.

*Clinical application of oVEMPs*

In the clinic, the oVEMP can be used like other vestibular function tests: to aid diagnosis, test the extent of a lesion or monitor disease progression or recovery. An advantage of oVEMPs over the main existing test of otolith function, subjective visual vertical/horizontal (SVV/H), is that the reflex remains abnormal even after central compensation has occurred. It is also more practical than eccentric rotation [27]. The oVEMP is best interpreted in light of other vestibular test results, and interpreted with caution when an oVEMP abnormality is the sole positive result, as false positives may be more common than isolated otolith abnormalities. Audiometric results are additionally important for interpreting AC oVEMPs, as conductive hearing loss can reduce or abolish the reflex.

Interpretation of oVEMP test abnormalities is generally similar to the cVEMP: an absent or attenuated reflex suggests loss of function, but does not indicate the specific cause, while an abnormally large reflex usually suggests the presence of a ‘third mobile window’ in the labyrinth, such as that caused by SCD. The range of normal amplitude values is large and extends down to zero, and thus amplitude alone is not always reliable in detecting vestibular loss. Instead, as responses are reasonably symmetric in normal subjects, comparison of the two sides can reveal significant asymmetry, with the abnormality usually on the smaller side, except in the less common cases of vestibular hyperfunction. An abnormality usually indicates a peripheral vestibular problem, but disorders affecting central vestibulo-ocular pathways can also affect the oVEMP. Latency prolongation with normal amplitude can also be associated with vestibular abnormalities, but is a less common finding associated primarily with diseases affecting nerve conduction along the VOR pathway, such as multiple sclerosis.

While many factors can have small attenuating effects on oVEMP amplitude, such as fatigue, alcohol intoxication or nystagmus, the reflex is generally very robust and not abolished by these conditions as long as a sufficient degree of gaze elevation is maintained during the test [28,29]. The procedure is short and does not elicit nausea, but requires some cooperation, such as elevation of the eyes and relaxation of the face without excessive blinking. It can therefore be performed in both the acute and chronic stages of vestibular illness, as long as the patient is well enough to look upwards and tolerate testing.

## oVEMPs in Typical Vestibular Diseases

*Superior Canal Dehiscence (SCD)*

Dehiscence of the superior semicircular canal (SCD) leads to sound- or pressure-induced vertigo via a third mobile window in the labyrinth [30]. Subjects usually complain of distinctive symptoms like autophony (loud perception of their own voice), as well as vertigo and oscillopsia induced by loud sounds at certain frequencies. The altered mechanical properties of the labyrinth lead to AC oVEMPs with high amplitudes and low thresholds [26,31-35], while low cVEMP thresholds have long been used in SCD diagnosis.

AC oVEMPs provide the best separation between normal subjects and patients with SCD [26,33], and the reflex normalizes after successful corrective surgery [35]. On average, oVEMPs show a 10-fold increase in n10 amplitude with little overlap with the normal range [26], though due to age effects patients should always be compared to age-matched normal controls [36]. The AC oVEMP has sensitivity and specificity of close to 100% depending on the stimuli and use of age-matched controls. BC stimulation also produces enhanced oVEMPs [31], though the separation between patients and controls is not always successful using BC vibration over the forehead (at Fz) [35]. In contrast, stimulation at the vertex (Cz) produces large oVEMPs in patients and very small responses in normal subjects [31,37].

SCD also produces characteristic changes in frequency tuning [37,36,38]. Normal oVEMPs and cVEMPs have tuning curves with optimal responses between about 400 and 1000 Hz to AC and between 100 and 250 Hz to BC [36,39-41]. In contrast, patients with SCD have much wider amplitude and threshold tuning curves [36,42,38]: AC oVEMP curves tend to shift upwards, while AC cVEMP curves tend to shift downwards [36]. Using a stimulus which only elicits a response in SCD patients, but not in normal subjects, is therefore a potential diagnostic tool [43].

oVEMPs may also be enlarged in symptomatic patients with ‘near-dehiscence’, as confirmed by thinning, but not frank dehiscence, of the bone roof over the superior semicircular canal at surgery [44]. Similarly, oVEMPs have proven to be useful for detecting patients with other related third mobile window labyrinth disorders, such as lateral and posterior semicircular canal dehiscence or large vestibular aqueduct syndrome [30,45]. These patients, too, show oVEMP normalization after successful surgery.

*Vestibular Neuritis*

Vestibular neuritis is characterised by acute onset of rotational vertigo and postural imbalance and is accompanied by spontaneous nystagmus, nausea or vomiting caused by loss of peripheral vestibular function [46]. Measurement of head impulses now makes it possible to test all three semicircular canals individually, especially since the introduction of the video head impulse test [47]. This has shown that in most cases of VN, afferents from both vestibular nerve portions are affected, or those from the superior nerve alone are affected [48,49]. Rarely, VN involves only the inferior nerve division, as shown by isolated posterior canal deficits [48,50,49].

oVEMPs are usually abnormal in VN, regardless of whether AC or BC stimuli are used [13,14,51,16,52,15,53]. The oVEMP abnormalities are most often associated with deficits in superior nerve horizontal and anterior canal fibres, and so these studies provide evidence that oVEMPs are probably mediated by superior nerve otolith fibres (which are mainly utricular), at least for the stimuli that have been tested thus far. In contrast, early studies showed that AC cVEMPs are usually normal in VN [54], providing supportive evidence that the AC cVEMP is produced by inferior nerve saccular afferents. This suggests that by using oVEMPs, AC cVEMPs and head impulses, we can test all five vestibular sub-organs individually.

A recent prospective study found that 55% of cases were combined superior and inferior VN, while 40% were superior VN (as defined by pathological oVEMPs, anterior canal and/or horizontal canal vHIT), and 5% were inferior VN (as defined by pathological cVEMPs and/or posterior canal vHIT) [49]. The deficits were often patchy, suggesting that the disease can be restricted to isolated parts of the inner ear. For example, selective VN affecting the lateral and superior semicircular canals has been described, based on the video head impulse test, absent calorics, but preserved oVEMPs and cVEMPs [55]. In the rare cases of VN where only the inferior division was involved, a reversed pattern of spared oVEMPs and affected cVEMPs was found [50]. Similarly, some patients with idiopathic sudden hearing loss also suffer from vertigo, suggesting involvement of the inferior division of the vestibular nerve, which is connected to the cochlear division via an anastomosis. In these patients, too, cVEMPs are more often affected than oVEMPs [56].

Magluilo et al. found that pathological oVEMPs in VN, suggesting utricular involvement, indicated a less favourable outcome with patients experiencing persisting vestibular symptoms at 3-month follow-up [49]. Similarly, Adamec et al. evaluated the recovery of patients after VN with VEMPs [57]. About 60% of their patients showed an improvement of AC oVEMPs after 6 months, while they found no change in cVEMPs. From a clinical perspective, the patients without oVEMP recovery, suggesting permanent utricular damage, had worse outcomes.  Accordingly, oVEMP recovery could potentially predict favourable outcome in patients with superior VN.

*Benign Paroxysmal Positional Vertigo*

Benign paroxysmal positional vertigo (BPPV) is characterized by brief episodes of rotational vertigo, typically triggered by a change in head position with respect to gravity [58]. The vertigo is caused by detached otoconia, which most often enter the posterior semicircular canal. Because of the anatomical connection to the semicircular canals, it has been assumed that the dislodged otoconia probably originate from the utricle rather than the saccule. Recent studies in patients with BPPV have found higher rates of abnormality for the oVEMP than the cVEMP, which is largely normal in BPPV, supporting this hypothesis [59-63].

*Vestibular Schwannoma*

Vestibular Schwannoma (VS) are benign tumors, which preferentially affect the inferior vestibular division of the vestibulocochlear nerve. Reliable monitoring of these usually slow-growing tumors is important, as ‘watchful waiting’ may be an appropriate strategy in some patients [64]. Along with measures of canal function, both oVEMPs and cVEMPs are often attenuated or absent in patients with tumors affecting the vestibular nerve or cerebellopontine angle [65-69]. Chiarovano at al. evaluated the clinical utility of VEMPs in patients with non-operated VS [65]. They found that BC oVEMPs were abnormal in 68% of the patients, while AC cVEMPs were abnormal in 65%, and the oVEMP was the only abnormal test in some of the patients. Similarly, Kinoshita et al. found that BC oVEMPs were abnormal in 69% of patients, and there was a significant correlation with caloric irrigation results, reflecting involvement of the superior vestibular nerve [66], similar to previous studies [69]. Lin et al. found that, in contrast to audiometry, oVEMPs and cVEMPs were significant predictors of tumor size [67], with tumors above 2 cm more likely to be associated with absent responses. Su et al. investigated the utility of oVEMPs and cVEMPs evoked by BC stimulation in diagnosing cerebellopontine angle meningioma and VS [68]. oVEMPs and cVEMPs had similar high rates of abnormality in the two diseases (above 80%).

*Menière's Disease*

The diagnosis of Menière's disease (MD) is currently based on clinical grounds, according to defined clinical criteria including recurrent attacks of spontaneous vertigo, hearing loss and tinnitus [70]. The disease fluctuates over time, with recurrent attacks and periods of quiescence, as well as permanent vestibular and hearing loss over time. In addition, there is considerable overlap with migraine [71]. In this context, the utility of oVEMPs has been investigated to help diagnose and classify MD patients.

Patients with MD have higher rates of attenuated or absent oVEMPs than normal subjects, increasing with advancing disease [72,73]. Winters et al. examined AC oVEMPs in MD patients [74], and found lower response rates with smaller amplitudes and higher thresholds compared to normal subjects. Interestingly, this effect was observed not only in the affected, but to a lesser extent also in the clinically unaffected ears. In contrast, BC oVEMPs show comparatively low rates of abnormality when recorded between attacks [75]. Taylor et al. compared AC and BC stimuli and found that the AC oVEMP was the most sensitive reflex, followed by the AC cVEMP, while BC oVEMPs and cVEMPs were less commonly affected. The same order of test abnormalities was found by Huang et al., who set out to localize hydrops formation in MD patients using oVEMPs, cVEMPs, audiometry and caloric irrigation [72]. From this finding the authors concluded that the order of loss may reflect the declining frequency of hydrops formation in temporal bone studies of MD patients (cochlea > saccule > utricle > semicircular canals)[76]. According to the anatomy of the labyrinth, MD may therefore affect the pars inferior (i.e. saccule and cochlea) more frequently than the pars superior (i.e. utricle and semicircular canals), consistent with anatomical studies.

Similar to cVEMPs [77], oVEMPs can also be augmented in MD [75,73,78]. Wen et al. recruited unilateral MD patients with either augmented or reduced oVEMPs (asymmetry >40 and <100%) in response to BC vibration [78], and found that augmented oVEMPs were seen more frequently in patients with early stage MD. Augmented responses might also be more likely when recorded during an attack. Manzari et al. found larger BC oVEMPs in patients with early MD during an acute attack compared to a quiescent period between attacks [75].

In order to distinguish patients with MD from normal subjects, tuning characteristics of the oVEMP stimulus frequency have attracted considerable attention. Compared to normal subjects, who have highest AC oVEMP amplitudes and lowest thresholds in response to tone bursts around 500 Hz [41,79], in MD patients the optimal stimulus frequency is shifted upwards to 1000 Hz [80,79,81]. At 500Hz, the oVEMP response rates are lower, amplitudes smaller, and thresholds higher than in subjects without MD [81,80]. This is similar to the pattern found for cVEMPs, which have previously been known to show altered tuning in MD [82]. Jerin et al. exploited this tuning effect by suggesting a 500Hz/1000Hz oVEMP amplitude ratio as a supportive indicator of MD [83]. In patients with definite MD, as defined by visualization of endolymphatic hydrops in MRI, they found significantly lower 500Hz/1000Hz amplitude ratios.

*Vestibular Migraine*

The overlap of signs and symptoms between vestibular migraine (VM) and MD has been well recognized in the literature [84]. Taylor et al. sought to disentangle the two diseases using VEMPs [79], and found that VM patients were indistinguishable from control subjects. A combination of test parameters, including the 500Hz/1000Hz cVEMP amplitude ratio, 500 Hz AC cVEMP asymmetry ratio and caloric testing differentiated MD from VM with sensitivity of 90% and specificity of 70%, compared to the caloric test alone with sensitivity of 75%. Similarly, Zuniga et al. found that 500 Hz AC oVEMPs were normal in VM patients and smaller in patients with MD, however, segregation of the two diseases occurred at the group, but not individual level [71].

*Central Vestibular Disorders*

Lesions in the brainstem may affect the crossed otolith-ocular pathways of oVEMPs, as well as the uncrossed otolith-spinal pathways of cVEMPs [85]. Studies in patients with acute brainstem lesions [86] and internuclear ophthalmoplegia (INO) [87,88,86] caused by stroke and multiple sclerosis (MS) suggest that the oVEMP pathways course along the medial longitudinal fasciculus (MLF) through the dorsomedial brainstem. The circuit remains functional independent of the cerebellum, as oVEMPs and cVEMPs tend to be preserved in isolated cerebellar lesions, but are often abolished in cerebellar lesions involving the brainstem [89].

Multiple sclerosis can cause not only abolished VEMPs, but also significant latency prolongation, as expected from the process of demyelination [85]. Therefore oVEMPs may be helpful to assess brainstem involvement in patients with MS [90-93], in particular of the MLF, which often becomes symptomatic as INO in these patients [87,88]. Gabelic et al. measured AC oVEMPs and cVEMPs in MS patients and found that patients with symptoms of brainstem involvement were more likely to show VEMP abnormalities than those without brainstem symptoms [91]. Therefore oVEMPs are likely to complement other evoked potentials in MS, like auditory brainstem responses, and may also detect silent lesions, though their comparative sensitivity is not yet known.

## Conclusions

Considerable research efforts have led to better understanding of oVEMP physiology and pathophysiology over the last years. With the typical stimulation and recording conditions used clinically, the underlying crossed reflex pathway has been shown to originate mainly in the superior vestibular nerve and thus provide a test of predominantly utricular function. The myogenic origin in the contralateral inferior oblique extraocular muscle has now been well established. As only little additional technical equipment is necessary, the test has made a successful transition from bench to bedside, and oVEMPs have become part of the test ensemble in most large balance centers.

**Table: Key Points**

- The oVEMP is a crossed excitatory reflex: originating mainly in the utricle, and coursing along the superior vestibular nerve to the contralateral inferior oblique eye muscle.

- Enlarged AC oVEMPs are an excellent screening parameter for superior canal dehiscence, with little overlap to normals.

- oVEMPs are helpful for diagnosing vestibular neuritis, indicating superior nerve otolith involvement.

- oVEMPs may be a useful monitoring tool for vestibular Schwannoma.

**Figure Legend**

Ocular vestibular evoked potentials in response to bone-conducted (BC) vibration and air-conducted (AC) sound. (A) Example of a patient with advanced left Menière's disease (MD) compared to a normal volunteer. Midline BC vibration of the forehead, which activates both ears, elicits a negative-positive (n10-p15) oVEMP underneath the left eye, originating from the healthy right ear. The oVEMP underneath the right eye, originating from the affected left ear, is abolished (shown by the black cross). (B) Example of a patient with left superior canal dehiscence (SCD). AC stimulation of the left SCD ear elicits a large oVEMP underneath the contralateral right eye. The same stimulus in a normal subject elicits a regular oVEMP on the right side. Note the reduced gain in part B to display the large SCD response.

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**Conflict of Interest**
The authors declare no conflict of interest.

**References of Importance**

• Of importance
•• Of major importance

[9] • Based on simultaneous needle and surface recordings, the study identified the inferior oblique as the eye muscle of origin of the oVEMP. The study for the first time characterized the single motor unit activity in human eye muscles during the vestibulo-ocular reflex.

[18] • This group has shown that oVEMPs can also successfully be recorded in children.

[26] •• The study identified oVEMPs in response to air-conducted sound as the best single-step screening test parameter for superior canal dehiscence.

[36] •• The authors carefully investigated the tuning characteristics of patients with superior canal dehiscence compared to normal subjects. Superior canal dehiscence led to a broadening of amplitude and threshold tuning curves. While AC oVEMPs tended to tune upwards, AC cVEMPs tended to tune downwards.

[42] • This case report was the first to show broadened tuning curves for BC oVEMPs in patients with SCD. The tuning returned to normal following surgery.

[43] • The study suggests oVEMPs with 4000 Hz AC or BC high-frequency stimulation as a specific indicator for superior canal dehiscence, which only elicits a response in patients with probable superior canal dehiscence, but not in normal subjects.

[49] • This large study tested all three semicircular canals and both otoliths using vHIT and VEMPs. They confirmed the finding that VN more often affects the superior nerve or whole nerve rather than the inferior nerve, and showed that the deficits are often only patchy.

[74] • This study compared amplitudes and thresholds of affected and unaffected ears in patients with different stages of MD. AC oVEMPs were nearly always present in normal control ears, and were smaller in the patients, especially in affected ears, but also on the unaffected side.

[66] • The authors tested AC cVEMPs, AC and BC oVEMPs, and calorics in patients with vestibular Schwannoma. They found that the latter three tests correlated with each other, but not with the cVEMP, suggesting that both AC and BC oVEMPs are mediated by the superior vestibular nerve.

[92] • The authors recorded AC oVEMPs and cVEMPs in patients with MS and found some cases of reflex absence and multiple cases of reflex delay, consistent with demyelination. The latencies were not significantly correlated with brainstem clinical or MRI findings, but were significantly related to disability score.

## References


1. Colebatch JG, Halmagyi GM, Skuse NF (1994) Myogenic potentials generated by a click-evoked vestibulocollic reflex. Journal of neurology, neurosurgery, and psychiatry 57 (2):190-197
2. Rosengren SM, McAngus Todd NP, Colebatch JG (2005) Vestibular-evoked extraocular potentials produced by stimulation with bone-conducted sound. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 116 (8):1938-1948. doi:10.1016/j.clinph.2005.03.019
3. Todd NP, Rosengren SM, Aw ST, Colebatch JG (2007) Ocular vestibular evoked myogenic potentials (OVEMPs) produced by air- and bone-conducted sound. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 118 (2):381-390. doi:10.1016/j.clinph.2006.09.025
4. Welgampola MS, Migliaccio AA, Myrie OA, Minor LB, Carey JP (2009) The human sound-evoked vestibulo-ocular reflex and its electromyographic correlate. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 120 (1):158-166. doi:10.1016/j.clinph.2008.06.020
5. Govender S, Rosengren SM, Colebatch JG (2009) The effect of gaze direction on the ocular vestibular evoked myogenic potential produced by air-conducted sound. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 120 (7):1386-1391. doi:10.1016/j.clinph.2009.04.017
6. Murnane OD, Akin FW, Kelly KJ, Byrd S (2011) Effects of stimulus and recording parameters on the air conduction ocular vestibular evoked myogenic potential. Journal of the American Academy of Audiology 22 (7):469-480. doi:10.3766/jaaa.22.7.7
7. Chihara Y, Iwasaki S, Ushio M, Murofushi T (2007) Vestibular-evoked extraocular potentials by air-conducted sound: another clinical test for vestibular function. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 118 (12):2745-2751. doi:10.1016/j.clinph.2007.08.005
8. Iwasaki S, McGarvie LA, Halmagyi GM, Burgess AM, Kim J, Colebatch JG, Curthoys IS (2007) Head taps evoke a crossed vestibulo-ocular reflex. Neurology 68 (15):1227-1229. doi:10.1212/01.wnl.0000259064.80564.21
9. Weber KP, Rosengren SM, Michels R, Sturm V, Straumann D, Landau K (2012) Single motor unit activity in human extraocular muscles during the vestibulo-ocular reflex. The Journal of physiology 590 (Pt 13):3091-3101. doi:10.1113/jphysiol.2011.226225
10. Rosengren SM, Colebatch JG, Straumann D, Weber KP (2013) Why do oVEMPs become larger when you look up? Explaining the effect of gaze elevation on the ocular vestibular evoked myogenic potential. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 124 (4):785-791. doi:10.1016/j.clinph.2012.10.012
11. Curthoys IS, Vulovic V, Sokolic L, Pogson J, Burgess AM (2012) Irregular primary otolith afferents from the guinea pig utricular and saccular maculae respond to both bone conducted vibration and to air conducted sound. Brain research bulletin 89 (1-2):16-21. doi:10.1016/j.brainresbull.2012.07.007
12. Zhu H, Tang X, Wei W, Maklad A, Mustain W, Rabbitt R, Highstein S, Allison J, Zhou W (2014) Input-output functions of vestibular afferent responses to air-conducted clicks in rats. Journal of the Association for Research in Otolaryngology : JARO 15 (1):73-86. doi:10.1007/s10162-013-0428-6
13. Curthoys IS, Iwasaki S, Chihara Y, Ushio M, McGarvie LA, Burgess AM (2011) The ocular vestibular-evoked myogenic potential to air-conducted sound; probable superior vestibular nerve origin. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 122 (3):611-616. doi:10.1016/j.clinph.2010.07.018

14. Govender S, Rosengren SM, Colebatch JG (2011) Vestibular neuritis has selective effects on air- and bone-conducted cervical and ocular vestibular evoked myogenic potentials. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 122 (6):1246-1255. doi:10.1016/j.clinph.2010.12.040
15. Iwasaki S, Chihara Y, Smulders YE, Burgess AM, Halmagyi GM, Curthoys IS, Murofushi T (2009) The role of the superior vestibular nerve in generating ocular vestibular-evoked myogenic potentials to bone conducted vibration at Fz. 2009/02/13 (3):588-593
16. Shin BS, Oh SY, Kim JS, Kim TW, Seo MW, Lee H, Park YA (2012) Cervical and ocular vestibular-evoked myogenic potentials in acute vestibular neuritis. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 123 (2):369-375. doi:10.1016/j.clinph.2011.05.029
17. Iwasaki S, Smulders YE, Burgess AM, McGarvie LA, MacDougall HG, Halmagyi GM, Curthoys IS (2008) Ocular vestibular evoked myogenic potentials to bone conducted vibration of the midline forehead at Fz in healthy subjects. 2008/07/22 (9):2135-2147
18. Chou CH, Hsu WC, Young YH (2012) Ocular vestibular-evoked myogenic potentials via bone-conducted vibration in children. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 123 (9):1880-1885. doi:10.1016/j.clinph.2012.02.059
19. Agrawal Y, Zuniga MG, Davalos-Bichara M, Schubert MC, Walston JD, Hughes J, Carey JP (2012) Decline in semicircular canal and otolith function with age. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 33 (5):832-839. doi:10.1097/MAO.0b013e3182545061
20. Nguyen KD, Welgampola MS, Carey JP (2010) Test-retest reliability and age-related characteristics of the ocular and cervical vestibular evoked myogenic potential tests. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 31 (5):793-802. doi:10.1097/MAO.0b013e3181e3d60e
21. Piker EG, Jacobson GP, McCaslin DL, Hood LJ (2011) Normal characteristics of the ocular vestibular evoked myogenic potential. Journal of the American Academy of Audiology 22 (4):222-230. doi:10.3766/jaaa.22.4.5
22. Rosengren SM, Govender S, Colebatch JG (2011) Ocular and cervical vestibular evoked myogenic potentials produced by air- and bone-conducted stimuli: comparative properties and effects of age. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 122 (11):2282-2289. doi:10.1016/j.clinph.2011.04.001
23. Tseng CL, Chou CH, Young YH (2010) Aging effect on the ocular vestibular-evoked myogenic potentials. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 31 (6):959-963. doi:10.1097/MAO.0b013e3181e8fb1a
24. Chang CM, Young YH, Cheng PW (2012) Age-related changes in ocular vestibular-evoked myogenic potentials via galvanic vestibular stimulation and bone-conducted vibration modes. Acta oto-laryngologica 132 (12):1295-1300. doi:10.3109/00016489.2012.708437
25. Colebatch JG, Govender S, Rosengren SM (2013) Two distinct patterns of VEMP changes with age. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 124 (10):2066-2068. doi:10.1016/j.clinph.2013.04.337
26. Janky KL, Nguyen KD, Welgampola M, Zuniga MG, Carey JP (2013) Air-conducted oVEMPs provide the best separation between intact and superior canal dehiscent labyrinths. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 34 (1):127-134. doi:10.1097/MAO.0b013e318271c32a

27. Valko Y, Hegemann SC, Weber KP, Straumann D, Bockisch CJ (2011) Relative diagnostic value of ocular vestibular evoked potentials and the subjective visual vertical during tilt and eccentric rotation. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 122 (2):398-404. doi:10.1016/j.clinph.2010.06.023
28. Rosengren SM, Weber KP, Hegemann SC, Roth TN (2014) The effect of alcohol on cervical and ocular vestibular evoked myogenic potentials in healthy volunteers. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 125 (8):1700-1708. doi:10.1016/j.clinph.2013.12.096
29. Lin BY, Young YH (2014) Effect of short-duration sleep deprivation on the vestibulo-ocular reflex system evaluated by ocular vestibular-evoked myogenic potential test. Acta oto-laryngologica 134 (7):698-703. doi:10.3109/00016489.2014.895039
30. Chien WW, Carey JP, Minor LB (2011) Canal dehiscence. Current opinion in neurology 24 (1):25-31. doi:10.1097/WCO.0b013e328341ef88
31. Manzari L, Burgess AM, McGarvie LA, Curthoys IS (2012) Ocular and cervical vestibular evoked myogenic potentials to 500 Hz fz bone-conducted vibration in superior semicircular canal dehiscence. Ear and hearing 33 (4):508-520. doi:10.1097/AUD.0b013e3182498c09
32. Thabet EM, Abdelkhalek A, Zaghloul H (2012) Superior semicircular canal dehiscence syndrome as assessed by oVEMP and temporal bone computed tomography imaging. European archives of oto-rhino-laryngology : official journal of the European Federation of Oto-Rhino-Laryngological Societies 269 (5):1545-1549. doi:10.1007/s00405-011-1893-3
33. Zuniga MG, Janky KL, Nguyen KD, Welgampola MS, Carey JP (2013) Ocular versus cervical VEMPs in the diagnosis of superior semicircular canal dehiscence syndrome. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 34 (1):121-126. doi:10.1097/MAO.0b013e31827136b0
34. Rosengren SM, Aw ST, Halmagyi GM, Todd NP, Colebatch JG (2008) Ocular vestibular evoked myogenic potentials in superior canal dehiscence. Journal of neurology, neurosurgery, and psychiatry 79 (5):559-568. doi:10.1136/jnnp.2007.126730
35. Welgampola MS, Myrie OA, Minor LB, Carey JP (2008) Vestibular-evoked myogenic potential thresholds normalize on plugging superior canal dehiscence. Neurology 70 (6):464-472. doi:10.1212/01.wnl.0000299084.76250.4a
36. Taylor RL, Bradshaw AP, Halmagyi GM, Welgampola MS (2012) Tuning characteristics of ocular and cervical vestibular evoked myogenic potentials in intact and dehiscent ears. Audiology & neuro-otology 17 (4):207-218. doi:10.1159/000336959
37. Taylor RL, Blaivie C, Bom AP, Holmeslet B, Pansell T, Brantberg K, Welgampola MS (2014) Ocular vestibular-evoked myogenic potentials (oVEMP) to skull taps in normal and dehiscent ears: mechanisms and markers of superior canal dehiscence. Experimental brain research Experimentelle Hirnforschung Experimentation cerebrale 232 (4):1073-1084. doi:10.1007/s00221-013-3782-z
38. Zhang AS, Govender S, Colebatch JG (2012) Tuning of the ocular vestibular evoked myogenic potential (oVEMP) to air- and bone-conducted sound stimulation in superior canal dehiscence. Experimental brain research Experimentelle Hirnforschung Experimentation cerebrale 223 (1):51-64. doi:10.1007/s00221-012-3240-3
39. Todd NP, Rosengren SM, Colebatch JG (2009) A utricular origin of frequency tuning to low-frequency vibration in the human vestibular system? Neuroscience letters 451 (3):175-180. doi:10.1016/j.neulet.2008.12.055
40. Chihara Y, Iwasaki S, Fujimoto C, Ushio M, Yamasoba T, Murofushi T (2009) Frequency tuning properties of ocular vestibular evoked myogenic potentials. Neuroreport 20 (16):1491-1495. doi:10.1097/WNR.0b013e3283329b4a

41. Park HJ, Lee IS, Shin JE, Lee YJ, Park MS (2010) Frequency-tuning characteristics of cervical and ocular vestibular evoked myogenic potentials induced by air-conducted tone bursts. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 121 (1):85-89. doi:10.1016/j.clinph.2009.10.003
42. Zhang AS, Govender S, Colebatch JG (2011) Superior canal dehiscence causes abnormal vestibular bone-conducted tuning. Neurology 77 (9):911-913. doi:10.1212/WNL.0b013e31822c6263
43. Manzari L, Burgess AM, McGarvie LA, Curthoys IS (2013) An indicator of probable semicircular canal dehiscence: ocular vestibular evoked myogenic potentials to high frequencies. Otolaryngology--head and neck surgery : official journal of American Academy of Otolaryngology-Head and Neck Surgery 149 (1):142-145. doi:10.1177/0194599813489494
44. Ward BK, Wenzel A, Ritzl EK, Gutierrez-Hernandez S, Della Santina CC, Minor LB, Carey JP (2013) Near-dehiscence: clinical findings in patients with thin bone over the superior semicircular canal. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 34 (8):1421-1428. doi:10.1097/MAO.0b013e318287efe6
45. Taylor RL, Bradshaw AP, Magnussen JS, Gibson WP, Halmagyi GM, Welgampola MS (2012) Augmented ocular vestibular evoked myogenic potentials to air-conducted sound in large vestibular aqueduct syndrome. Ear and hearing 33 (6):768-771. doi:10.1097/AUD.0b013e31825ce613
46. Jeong SH, Kim HJ, Kim JS (2013) Vestibular neuritis. Seminars in neurology 33 (3):185-194. doi:10.1055/s-0033-1354598
47. MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS (2009) The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology 73 (14):1134-1141. doi:10.1212/WNL.0b013e3181bacf85
48. Aw ST, Fetter M, Cremer PD, Karlberg M, Halmagyi GM (2001) Individual semicircular canal function in superior and inferior vestibular neuritis. Neurology 57 (5):768-774
49. Magliulo G, Gagliardi S, Ciniglio Appiani M, Iannella G, Re M (2014) Vestibular neurolabyrinthitis: a follow-up study with cervical and ocular vestibular evoked myogenic potentials and the video head impulse test. The Annals of otology, rhinology, and laryngology 123 (3):162-173. doi:10.1177/0003489414522974
50. Kim JS, Kim HJ (2012) Inferior vestibular neuritis. Journal of neurology 259 (8):1553-1560. doi:10.1007/s00415-011-6375-4
51. Lin CM, Young YH (2011) Identifying the affected branches of vestibular nerve in vestibular neuritis. Acta oto-laryngologica 131 (9):921-928. doi:10.3109/00016489.2011.573504
52. Oh SY, Kim JS, Yang TH, Shin BS, Jeong SK (2013) Cervical and ocular vestibular-evoked myogenic potentials in vestibular neuritis: comparison between air- and bone-conducted stimulation. Journal of neurology 260 (8):2102-2109. doi:10.1007/s00415-013-6953-8
53. Manzari L, Tedesco A, Burgess AM, Curthoys IS (2010) Ocular vestibular-evoked myogenic potentials to bone-conducted vibration in superior vestibular neuritis show utricular function. Otolaryngology--head and neck surgery : official journal of American Academy of Otolaryngology-Head and Neck Surgery 143 (2):274-280. doi:10.1016/j.otohns.2010.03.020
54. Murofushi T, Halmagyi GM, Yavor RA, Colebatch JG (1996) Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis. An indicator of inferior vestibular nerve involvement? Archives of otolaryngology--head & neck surgery 122 (8):845-848
55. Magliulo G, Gagliardi S, Ciniglio Appiani M, Iannella G, Gagliardi M (2012) Selective vestibular neurolabyrinthitis of the lateral and superior semicircular canal ampulla and ampullary nerves. The Annals of otology, rhinology, and laryngology 121 (10):640-644

56. Fujimoto C, Egami N, Kinoshita M, Sugasawa K, Yamasoba T, Iwasaki S (2014) Involvement of vestibular organs in idiopathic sudden hearing loss with vertigo: An analysis using oVEMP and cVEMP testing. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology. doi:10.1016/j.clinph.2014.07.028
57. Adamec I, Skoric MK, Handzic J, Barusic AK, Bach I, Gabelic T, Habek M (2014) The role of cervical and ocular vestibular-evoked myogenic potentials in the follow-up of vestibular neuritis. Clinical EEG and neuroscience 45 (2):129-136. doi:10.1177/1550059413483452
58. Kim JS, Zee DS (2014) Clinical practice. Benign paroxysmal positional vertigo. The New England journal of medicine 370 (12):1138-1147. doi:10.1056/NEJMcp1309481
59. Bremova T, Bayer O, Agrawal Y, Kremmyda O, Brandt T, Teufel J, Strupp M (2013) Ocular VEMPs indicate repositioning of otoconia to the utricle after successful liberatory maneuvers in benign paroxysmal positioning vertigo. Acta oto-laryngologica 133 (12):1297-1303. doi:10.3109/00016489.2013.829922
60. Lee JD, Park MK, Lee BD, Lee TK, Sung KB, Park JY (2013) Abnormality of cervical vestibular-evoked myogenic potentials and ocular vestibular-evoked myogenic potentials in patients with recurrent benign paroxysmal postitional vertigo. Acta oto-laryngologica 133 (2):150-153. doi:10.3109/00016489.2012.723823
61. Nakahara H, Yoshimura E, Tsuda Y, Murofushi T (2013) Damaged utricular function clarified by oVEMP in patients with benign paroxysmal positional vertigo. Acta oto-laryngologica 133 (2):144-149. doi:10.3109/00016489.2012.720030
62. Seo T, Saka N, Ohta S, Sakagami M (2013) Detection of utricular dysfunction using ocular vestibular evoked myogenic potential in patients with benign paroxysmal positional vertigo. Neuroscience letters 550:12-16. doi:10.1016/j.neulet.2013.06.041
63. Singh NK, Barman A (2014) Efficacy of Ocular Vestibular-Evoked Myogenic Potential in Identifying Posterior Semicircular Canal Benign Paroxysmal Positional Vertigo. Ear and hearing. doi:10.1097/AUD.0000000000000097
64. Backous DD, Pham HT (2007) Guiding patients through the choices for treating vestibular schwannomas: balancing options and ensuring informed consent. Otolaryngologic clinics of North America 40 (3):521-540, viii-ix. doi:10.1016/j.otc.2007.03.004
65. Chiarovano E, Darlington C, Vidal PP, Lamas G, de Waele C (2014) The role of cervical and ocular vestibular evoked myogenic potentials in the assessment of patients with vestibular schwannomas. PloS one 9 (8):e105026. doi:10.1371/journal.pone.0105026
66. Kinoshita M, Iwasaki S, Fujimoto C, Inoue A, Egami N, Chihara Y, Ushio M, Yamasoba T (2013) Ocular vestibular evoked myogenic potentials in response to air-conducted sound and bone-conducted vibration in vestibular schwannoma. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 34 (7):1342-1348. doi:10.1097/MAO.0b013e31828d6539
67. Lin KL, Chen CM, Wang SJ, Young YH (2014) Correlating vestibular schwannoma size with vestibular-evoked myogenic potential results. Ear and hearing 35 (5):571-576. doi:10.1097/AUD.0000000000000046
68. Su CH, Chen CM, Young YH (2013) Differentiating cerebellopontine angle meningioma from schwannoma using caloric testing and vestibular-evoked myogenic potentials. Journal of the neurological sciences 335 (1-2):155-159. doi:10.1016/j.jns.2013.09.020
69. Iwasaki S, Murofushi T, Chihara Y, Ushio M, Suzuki M, Curthoys IS, Yamasoba T (2010) Ocular vestibular evoked myogenic potentials to bone-conducted vibration in vestibular schwannomas. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 31 (1):147-152. doi:10.1097/MAO.0b013e3181c0e670

70. Committee on Hearing and Equilibrium guidelines for the diagnosis and evaluation of therapy in Meniere's disease. American Academy of Otolaryngology-Head and Neck Foundation, Inc (1995). Otolaryngology--head and neck surgery : official journal of American Academy of Otolaryngology-Head and Neck Surgery 113 (3):181-185
71. Zuniga MG, Janky KL, Schubert MC, Carey JP (2012) Can vestibular-evoked myogenic potentials help differentiate Meniere disease from vestibular migraine? Otolaryngology--head and neck surgery : official journal of American Academy of Otolaryngology-Head and Neck Surgery 146 (5):788-796. doi:10.1177/0194599811434073
72. Huang CH, Wang SJ, Young YH (2011) Localization and prevalence of hydrops formation in Meniere's disease using a test battery. Audiology & neuro-otology 16 (1):41-48. doi:10.1159/000312199
73. Taylor RL, Wijewardene AA, Gibson WP, Black DA, Halmagyi GM, Welgampola MS (2011) The vestibular evoked-potential profile of Meniere's disease. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 122 (6):1256-1263. doi:10.1016/j.clinph.2010.11.009
74. Winters SM, Campschroer T, Grolman W, Klis SF (2011) Ocular vestibular evoked myogenic potentials in response to air-conducted sound in Meniere's disease. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 32 (8):1273-1280. doi:10.1097/MAO.0b013e31822e5ac9
75. Manzari L, Tedesco AR, Burgess AM, Curthoys IS (2010) Ocular and cervical vestibular-evoked myogenic potentials to bone conducted vibration in Meniere's disease during quiescence vs during acute attacks. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 121 (7):1092-1101. doi:10.1016/j.clinph.2010.02.003
76. Okuno T, Sando I (1987) Localization, frequency, and severity of endolymphatic hydrops and the pathology of the labyrinthine membrane in Meniere's disease. The Annals of otology, rhinology, and laryngology 96 (4):438-445
77. Young YH, Huang TW, Cheng PW (2003) Assessing the stage of Meniere's disease using vestibular evoked myogenic potentials. Archives of otolaryngology--head & neck surgery 129 (8):815-818. doi:10.1001/archotol.129.8.815
78. Wen MH, Cheng PW, Young YH (2012) Augmentation of ocular vestibular-evoked myogenic potentials via bone-conducted vibration stimuli in Meniere disease. Otolaryngology--head and neck surgery : official journal of American Academy of Otolaryngology-Head and Neck Surgery 146 (5):797-803. doi:10.1177/0194599811433982
79. Taylor RL, Zagami AS, Gibson WP, Black DA, Watson SR, Halmagyi MG, Welgampola MS (2012) Vestibular evoked myogenic potentials to sound and vibration: characteristics in vestibular migraine that enable separation from Meniere's disease. Cephalalgia : an international journal of headache 32 (3):213-225. doi:10.1177/0333102411434166
80. Sandhu JS, Low R, Rea PA, Saunders NC (2012) Altered frequency dynamics of cervical and ocular vestibular evoked myogenic potentials in patients with Meniere's disease. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 33 (3):444-449. doi:10.1097/MAO.0b013e3182488046
81. Winters SM, Berg IT, Grolman W, Klis SF (2012) Ocular vestibular evoked myogenic potentials: frequency tuning to air-conducted acoustic stimuli in healthy subjects and Meniere's disease. Audiology & neuro-otology 17 (1):12-19. doi:10.1159/000324858
82. Rauch SD, Zhou G, Kujawa SG, Guinan JJ, Herrmann BS (2004) Vestibular evoked myogenic potentials show altered tuning in patients with Meniere's disease. Otology & neurotology : official publication of the American Otological Society, American Neurotology Society [and] European Academy of Otology and Neurotology 25 (3):333-338

83. Jerin C, Berman A, Krause E, Ertl-Wagner B, Gurkov R (2014) Ocular vestibular evoked myogenic potential frequency tuning in certain Meniere's disease. Hearing research 310:54-59. doi:10.1016/j.heares.2014.02.001
84. Shepard NT (2006) Differentiation of Meniere's disease and migraine-associated dizziness: a review. Journal of the American Academy of Audiology 17 (1):69-80
85. Shimizu K, Murofushi T, Sakurai M, Halmagyi M (2000) Vestibular evoked myogenic potentials in multiple sclerosis. Journal of neurology, neurosurgery, and psychiatry 69 (2):276-277
86. Oh SY, Kim JS, Lee JM, Shin BS, Hwang SB, Kwak KC, Kim C, Jeong SK, Kim TW (2013) Ocular vestibular evoked myogenic potentials induced by air-conducted sound in patients with acute brainstem lesions. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 124 (4):770-778. doi:10.1016/j.clinph.2012.09.026
87. Rosengren SM, Colebatch JG (2011) Ocular vestibular evoked myogenic potentials are abnormal in internuclear ophthalmoplegia. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 122 (6):1264-1267. doi:10.1016/j.clinph.2010.10.040
88. Rosengren SM, Nogajski JH, Cremer PD, Colebatch JG (2007) Delayed vestibular evoked responses to the eyes and neck in a patient with an isolated brainstem lesion. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 118 (9):2112-2116. doi:10.1016/j.clinph.2007.05.068
89. Su CH, Young YH (2011) Differentiating cerebellar and brainstem lesions with ocular vestibular-evoked myogenic potential test. European archives of oto-rhino-laryngology : official journal of the European Federation of Oto-Rhino-Laryngological Societies 268 (6):923-930. doi:10.1007/s00405-010-1463-0
90. Gabelic T, Krbot M, Sefer AB, Isgum V, Adamec I, Habek M (2013) Ocular and cervical vestibular evoked myogenic potentials in patients with multiple sclerosis. Journal of clinical neurophysiology : official publication of the American Electroencephalographic Society 30 (1):86-91. doi:10.1097/WNP.0b013e31827eda0c
91. Gabelic T, Krbot Skoric M, Adamec I, Barun B, Zadro I, Habek M (2014) The vestibular evoked myogenic potentials (VEMP) score: a promising tool for evaluation of brainstem involvement in multiple sclerosis. European journal of neurology : the official journal of the European Federation of Neurological Societies. doi:10.1111/ene.12557
92. Gazioglu S, Boz C (2012) Ocular and cervical vestibular evoked myogenic potentials in multiple sclerosis patients. Clinical neurophysiology : official journal of the International Federation of Clinical Neurophysiology 123 (9):1872-1879. doi:10.1016/j.clinph.2012.01.022
93. Ivankovic A, Nesek Madaric V, Starcevic K, Krbot Skoric M, Gabelic T, Adamec I, Habek M (2013) Auditory evoked potentials and vestibular evoked myogenic potentials in evaluation of brainstem lesions in multiple sclerosis. Journal of the neurological sciences 328 (1-2):24-27. doi:10.1016/j.jns.2013.02.005